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FILE 'BIOSIS' ENTERED AT 20:30:03 ON 22 MAY 2008

FILE 'CAPLUS' ENTERED AT 20:30:03 ON 22 MAY 2008

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L1 672 (S6 KINASE) (2A) (INHIBITOR OR INHIBIT OR INHIBITING OR INHIBITED OR INHIBITION OR MODULATOR OR MODULATE OR MODULATING OR MODULATED OR MODULATION)

=> S L1 (6A) (treat or treatment treated or treating or administration or administer or administered or administering or therapy or therapeutic or medication or remedy or in vivo)
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L3 6 DUPLICATE REMOVE L2 (0 DUPLICATES REMOVED)

=> d l3 1-6 bib ab

L3 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:1302278 CAPLUS
DN 146:268807
TI BI-D1870 is a specific inhibitor of the p90 RSK (ribosomal S6 kinase) isoforms in vitro and in vivo
AU Sapkota, Gopal P.; Cummings, Lorna; Newell, Felicity S.; Armstrong, Christopher; Bain, Jennifer; Frodin, Morten; Grauert, Matthias; Hoffmann, Matthias; Schnapp, Gisela; Steegmaier, Martin; Cohen, Philip; Alessi, Dario R.
CS MRC Protein Phosphorylation Unit, School of Life Sciences, MSI/WTB Complex, University of Dundee, Dundee, DD1 5EH, UK
SO Biochemical Journal (2007), 401(1), 29-38
CODEN: BIJOAK; ISSN: 0264-6021
PB Portland Press Ltd.
DT Journal
LA English

AB Hormones and growth factors induce the activation of a number of protein kinases that belong to the AGC subfamily, including isoforms of PKA, protein kinase B (also known as Akt), PKC, S6K p70 (ribosomal S6 kinase), RSK (p90 ribosomal S6 kinase) and MSK (mitogen- and stress-activated protein kinase), which then mediate many of the physiol. processes that are regulated by these extracellular agonists. It can be difficult to assess the individual functions of each AGC kinase because their substrate specificities are similar. Here we describe the small mol. BI-D1870, which inhibits RSK1, RSK2, RSK3 and RSK4 in vitro with an IC50 of 10-30 nM, but does not significantly inhibit ten other AGC kinase members and over 40 other protein kinases tested at 100-fold higher concns. BI-D1870 is cell permeant and prevents the RSK-mediated phorbol ester- and EGF (epidermal growth factor)-induced phosphorylation of glycogen synthase kinase-3β and LKB1 in human embryonic kidney 293 cells and Rat-2 cells. In contrast, BI-D1870 does not affect the agonist-triggered phosphorylation of substrates for six other AGC kinases. Moreover, BI-D1870 does not suppress the phorbol ester- or EGF-induced phosphorylation of CREB (cAMP-response-element-binding protein), consistent with the genetic evidence indicating that MSK, and not RSK, isoforms mediate the mitogen-induced phosphorylation of this transcription factor.
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:381280 CAPLUS
DN 144:410248
TI Regulation of gluconeogenesis-associated genes by controling phosphorylation level of hepatocyte nuclear factor 4α by ribosomal S6 kinase B
IN Doi, Hirofumi; Shozaki, Yuka; Kudo, Gen
PA Daiichi Pharmaceutical Co., Ltd., Japan
SO PCT Int. Appl., 77 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006043701	A1	20060427	WO 2005-JP19518	20051024
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	JP 2008017701	A	20080131	JP 2004-308602	20041022
PRAI	JP 2004-308602	A	20041022		

AB Regulation method of gluconeogenesis-associated genes (phosphoenolpyrvate calboxykinase gene) by controlling phosphorylation level of transcription factor HNF-4α (hepatocyte nuclear factor 4α) by RSKB (ribosomal S6 kinase B) has been developed. Promotion of phosphorylation of HNF-4α by RSKB is used in the transcriptional activation of the phosphoenolpyruvate carboxykinase gene. The inhibition of RSKB-driven phosphorylation of HNF-4α by their specific antibodies or RSKB

inhibitors is used in the transcriptional repression of the phosphoenolpyruvate carboxykinase gene. This inhibitory mechanism is designed to be applied to the regulation of gluconeogenesis and therapy and prevention of diabetes. The cellular assay system of RSKB-driven phosphorylation of HNF-4α has been developed for the screening of inhibitors that can be the drugs for therapy and prevention of diabetes. The DNAs encoding RSKB and HNF-4α and the expression vectors containing them are provided in such assay kit for cell transformation.

RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:84678 CAPLUS
DN 144:347497
TI In vivo inhibition of nitric oxide synthase impairs upregulation of contractile protein mRNA in overloaded plantaris muscle
AU Sellman, Jeff E.; DeRuisseau, Keith C.; Betters, Jenna L.; Lira, Vitor A.; Soltow, Quinlyn A.; Selsby, Joshua T.; Criswell, David S.
CS Center for Exercise Science, University of Florida, Gainesville, FL, USA
SO Journal of Applied Physiology (2006), 100(1), 258-265
CODEN: JAPHEV; ISSN: 8750-7587
PB American Physiological Society
DT Journal
LA English
AB Inhibition of nitric oxide synthase (NOS) activity in vivo impedes hypertrophy in the overloaded rat plantaris. We investigated the mechanism for this effect by examining early events leading to muscle growth following 5 or 12 days of functional overload. Male Sprague-Dawley rats (.apprx.350 g) were randomly divided into three treatment groups: control, NG-nitro-L-arginine Me ester (L-NAME; 90 mg·kg-1·day-1), and 1-(2-trifluoromethyl-phenyl)-imidazole (TRIM; 10 mg·kg-1·day-1). Unilateral removal of synergists induced chronic overload (OL) of the right plantaris. Sham surgery performed on the left hindlimb served as a normally loaded control. L-NAME and TRIM treatments prevented OL-induced skeletal α-actin and type I (slow) myosin heavy chain mRNA expression at 5 days. Conversely, neither L-NAME nor TRIM affected hepatocyte growth factor or VEGF mRNA responses to OL at 5 days. However, OL induction of IGF-I and mechanogrowth factor mRNA was greater ($P < 0.05$) in the TRIM group compared with the controls. Furthermore, the phosphorylated-to-total p70 S6 kinase ratio was higher in OL muscle from NOS-inhibited groups, compared with control OL. At 12 days of OL, the cumulative proliferation of plantaris satellite cells was assessed by s.c. implantation of time release 5'-bromo-2'-deoxyuridine pellets during the OL-inducing surgeries. Although OL caused a fivefold increase in the number of mitotically active (5'-bromo-2'-deoxyuridine pos.) sublaminar nuclei, this was unaffected by concurrent NOS inhibition. Therefore, NOS activity may provide neg. feedback control of IGF-I/p70 S6 kinase signaling during muscle growth. Moreover, NOS activity may be involved in transcriptional regulation of skeletal α-actin and type I (slow) myosin heavy chain during functional overload.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:182924 CAPLUS
DN 142:254624
TI Inhibition of S6 kinase activity for the treatment of insulin resistance
IN Auwerx, Johan; Frigerio, Francesca; Fumagalli, Stefano; Kozma, Sara; Picard, Frederic; Sticker-Jantscheff, Melanie; Thomas, George; Um, Sung Hee; Watanabe, Mitsuhiro
PA Novartis Forschungsstiftung, Zweigniederlassung Friedrich Miescher

Institute for Biomedical Research, Switz.
SO Patent Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005019829	A1	20050303	WO 2004-EP9368	20040820
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1658504	A1	20060524	EP 2004-764350	20040820
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007503203	T	20070222	JP 2006-523617	20040820
US 20070191259	A1	20070816	US 2007-568637	20070322
PRAI US 2003-497226P	P	20030822		
WO 2004-EP9368	W	20040820		

AB The invention provides screening methods for agents effective in treating insulin resistance through specific inhibition of S6 kinase 1 activity. Also provided are methods of treating insulin resistance by administering an effective amount of an inhibitor specific for S6 kinase 1.
The inhibitor may be e.g. an antibody.
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:355107 CAPLUS
DN 140:368702
TI Modulation of S6 kinase 1 activity for the treatment of obesity and screening for antiobesity drugs
IN Frigerio, Francesca; Fumagalli, Stefano; Kozma, Sara C.; Sticker-Jantscheff, Melanie; Thomas, George; Um, Sung Hee
PA Novartis Forschungsstiftung, Zweigniederlassung Friedrich Miescher Institute for Biomedical Research, Switz.
SO PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004035815	A1	20040429	WO 2003-EP11554	20031017
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,				

FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	AU 2003282035	A1	20040504	AU 2003-282035	20031017
	EP 1556505	A1	20050727	EP 2003-773650	20031017
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	JP 2006502744	T	20060126	JP 2005-501296	20031017
	US 20070053910	A1	20070308	US 2006-531515	20060616
PRAI	GB 2002-24338	A	20021018		
	US 2003-497227P	P	20030822		
	WO 2003-EP11554	W	20031017		

AB This invention provides screening methods for agents effective in treating obesity through specific inhibition of S6 kinase 1 activity. Also provided are methods of treating obesity by administering an effective amount of an inhibitor specific for S6 kinase 1. A double stranded RNA for use as a medicament is provided.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:142886 CAPLUS
DN 140:177851
TI Diagnosis and treatment of diseases arising from defects in the tuberous sclerosis pathway by detecting and inhibiting increased S6 kinase activity
IN Guan, Kun-liang
PA The Regents of the University of Michigan, USA
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004014222	A2	20040219	WO 2003-US25283	20030812
	WO 2004014222	A3	20040521		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2495085	A1	20040219	CA 2003-2495085	20030812
	AU 2003259803	A1	20040225	AU 2003-259803	20030812
	AU 2003259803	B2	20070802		
	EP 1546369	A2	20050629	EP 2003-785242	20030812
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	CN 1688714	A	20051026	CN 2003-824037	20030812
	JP 2005535332	T	20051124	JP 2004-528088	20030812
PRAI	US 2002-402718P	P	20020812		
	US 2003-639263	A	20030812		
	WO 2003-US25283	W	20030812		

AB The present invention relates to compns. and methods for identifying abnormalities in TSC signaling pathways. In particular, the present invention relates to methods of diagnosing and treating disorders such as tuberous sclerosis, which are caused by mutations in the TSC genes. The

present invention further relates to methods and compns. for treating cancers mediated by TSC signaling disorders. A method of detecting increased S6 kinase activity in a subject comprises (a) providing a biol. sample from a subject; and (b) detecting the presence or absence of increased S6 kinase activity in the sample. An inhibitor of S6 kinase is administered to treat tuberous sclerosis. Expts. conducted during the course of development of the present invention demonstrated that TSC1-TSC2 complex inhibits the phosphorylation of S6K and 4E-BP1. The data show that TSC1-TSC2 exerts its effects through mTOR to regulate the activity of S6K and 4E-BP1. Further expts. demonstrated that the function of TSC1-TSC2 is neg. regulated by Akt-dependent phosphorylation in response to treatment with insulin and that the ability of TSC2 to inhibit S6K correlates with its tumor suppressor function.